the production of thyroid hormone is blocked because the biphenyls somehow mimic the hormone.

If these hypothetical relationships are the case, it would be prudent to follow children born in Michigan after the PBB release to determine whether thyroid abnormalities have been a problem. In addition, variations in growth or maturation of these children should be examined if there is a way to reconstruct past growth patterns at this late date. (These children are now in their early twenties.)

The evidence collected by Colborn et al. (*1*) suggests that the follow-up of children exposed *in utero* and postnatally to PBBs should include indicators of reproductive and developmental problems in this group and in their offspring, that is, in the next generation. In the study of PBB-exposed workers in New Jersey, we attempted to examine reproductive outcome but were unable to show differences between exposed and unexposed workers and their spouses because of too few pregnancies in this small cohort of workers (36 men in the PBB cohort) (*4*). However, it would be a good idea to follow the children of former PBB workers, if they can be identified, to ascertain their reproductive histories. Again, we find that follow-up of mobile U.S. populations is difficult. Thus, it is necessary to alert the scientific and medical communities to the potential for these problems in the event that their cause is not readily discernible or that their cause becomes a confounder in other studies of environmental toxicity.

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Erratum

In the Correspondence by Mushak and Crocetti (Response: Accuracy, Arsenic, and Cancer) published in EHP in Volume 104, Number 10, 1996, two sentences were printed incorrectly. The correct sentences are as follows: "Neither can Slayton et al. (*1*). Slayton et al. note that the Pyles and Woolsen report (*10*) did not include data on rice."